BRITISH JOURNAL OF
TUBERCULOSIS
AND DISEASES OF THE CHEST

Vol. XXXVII. July-October, 1943 Nos. 3-4

EDITORIAL

Our present issue is a combined one for various reasons, some not within our control. We trust that readers who felt the need of suitable literature in the summer, will feel rewarded in their autumn evenings by the miscellaneous provisions of a double number. To select for comment here is a task somewhat invidious; but, to make a personal choice as the privilege of an editor, it seems that the report included of an unusual complication of gold therapy raises again the troublesome problem of the place which gold really ought to occupy in the treatment of phthisis. That gold has lost much or almost all of its former popularity is not necessarily reason for regarding it as useless. There is a fashion in drugs, as in other things, all the more likely to bring neglect when initial enthusiasm has subsided in disappointment. This has happened many times in therapeutics; even prontosil was known and tried many years ago, yet nothing came of it at the time.

We are not supposing that in tuberculosis gold has a specific action in the least degree comparable with that of sulphonamides in pyogenic infections. All the evidence is against any such belief. Møllgaard's original work was marred by the use of devitalised strains and inadequate controls, as well as by neglect of statistical principles in the assessment of results. Subsequent observers, who tried to show curative effects in animals, invariably failed to do so when such errors were avoided in the design and interpretation of their experiments. The problem now—if there is a problem, which we believe to be the case—has moved from the question of specific cure to the nature and importance of the action of gold upon tuberculous tissues and whether there is ever any benefit to be gained for the patient.

The animal experiments which clearly showed that gold had no curative value also showed that it undoubtedly changed the tissue reactions to tubercles, chiefly in the direction of increasing their vascularity and enhancing the proliferative reaction—changes which might well be expected to have some beneficial application. Such effects are, of course, not peculiar to gold; they are seen, in more or less degree, in other infections from other agents. In general they are common to agents which, in virtue of molecular size or composition, exert a " shock-like " action upon the body. On this view gold treatment in the tuberculous is the equivalent of protein shock therapy in other chronic infections.

Unlike substances for producing such reactions which have no dangerous toxic effects in themselves apart from the shock-like effect, gold is also highly

toxic in virtue of its properties as a heavy metal. Some years ago recognition of this fact restricted the doses, regarded as reasonably safe, to a limit at which the capacity of the drug to produce any change in tuberculous tissues became doubtful. We have to add to this objection the possibility that even small amounts may sensitise the individual in a dangerous way; that there are, in fact, two risks—that run by the sensitised person and that of true cumulative poisoning, the ordinary metallic risk. The same double danger is seen with other heavy metals—mercury, for instance—though it almost looks as though, with gold, the sensitisation risk is greater than for others. Sunderlin's finding of 694 complications in 1,095 courses of gold injections, given for chronic arthritis, adds point to this opinion.

It certainly looks as though the restrictions which we are bound to observe with gold treatment place a very narrow limit upon its value in phthisis, even if it cannot be denied that the amounts employed may affect tuberculous tissues in a way that can be beneficial. Viewing the matter so, the future for gold looks far from promising; and it would certainly take many years of close work to show that any limited advantages are worth the risks in the average run of patients for whom it is still employed. For the patient in a corner, —as Ligden's patient was—unsuited for other measures, and yet offering some prospect of such measures becoming available in time if a present deterioration can be stopped, the situation may be regarded as different. Here there is everything to gain, nothing to lose. A situation in which the individual is likely to die from his phthisis can hardly be said to contra-indicate the use of gold provided that the disease is itself not likely to be adversely affected. Admittedly this places gold among the list of desperate remedies. Perhaps that really is its true place in therapeutics.

C. H.

GENERAL ARTICLES

THORACIC INJURIES: THE RÔLE OF THE GENERAL SURGEON IN THE FORWARD AREA*

By C. PRICE THOMAS.
From the Westminster Hospital and the Thoracic Unit, Horton Hospital, Epsom.

For the purposes of this paper the term general surgeon refers to the surgeon who is not conversant with the technique of intrathoracic manipulation, or, alternatively, the thoracic surgeon when so placed that facilities for complete thoracic operations are not available, especially a competent radiological service. Forward area is meant to refer to forward operating units where operative facilities are not the elaborate ones found in relatively stationary areas.

Experience in Libya has conclusively shown that there is no room for the specialist in the forward area. Unless there is a totally unexpected change in the present technique of warfare the thoracic unit will be concerned in the main with the late treatment of chest injuries—that is, with the late complications.

Many factors affect the speed with which casualties reach a forward operating unit. Perhaps the most important are the speed with which the action presses forward and climatic conditions at the time. Both these factors are of prime consideration in the first-aid treatment and transport of cases, with which we are not concerned at the moment; but they are of importance also in the present connection, as they affect most vitally the further course of thoracic injuries.

The more serious thoracic injuries often prove fatal before there is any possibility of removal to an operating unit, and it is probable that the proportion of dead to wounded may be greater in this rapid form of warfare than in the older, more static, form. The cause of death in these cases, apart from shock and hæmorrhage, potent enough in themselves, is severe interference with the cardio-respiratory mechanism. These factors continue to operate as a cause of death during the first forty-eight hours after receipt of the injury; if the patient survives this period, the cause of death is generally a direct result of superimposed infection.

Reports from Libya show that marked conservatism in the treatment of chest wounds has been attended with good results, and doubtless under the circumstances maintaining in this theatre this course has proved to have been the correct one. We should, however, be cautious in accepting this course as of universal application, especially as it is in marked conflict with experience

* Read before The Society of Thoracic Surgeons of Great Britain and Ireland in opening a discussion at their Annual Meeting, 1943.

gained in treating air-raid casualties and casualties during battle practice in this country. It is more than probable that climatic conditions, the relative sterility of the air, and the character of the country, in that the soil is uncultivated, had a considerable influence on the relative immunity of wounds to infection. Consequently it would probably be unwise to take Libyan conditions as a standard for treatment of wounds received in heavily cultivated areas such as those of Europe.

The aim of treatment is to decrease first the mortality rate and then the morbidity rate.

The chief function of the forward operating unit will be the decrease of the mortality rate, but at the same time both the early and late morbidity should not be lost sight of.

Shock is a variable factor in chest injuries; when present it is often associated with and sometimes masks a serious interference with a patient's cardio-respiratory reserve. Severity of shock usually is in proportion to the degree of tissue damage, and here, as elsewhere, a clean through-and-through gunshot wound is often accompanied by little or no shock, whereas laceration produced by the explosive fracture of ribs hit by a rifle bullet or by a fragment of shell, or a severe crush, may produce a serious degree of shock. In many cases the simple anti-shock remedies of warmth, both externally and internally, of hot drinks with morphia to control pain will be enough. These measures, however, are not of themselves sufficient if the shock is severe, and resort to plasma or whole-blood transfusions should not be too long delayed. Delay induces damage in the capillary bed, which makes later efforts at resuscitation quite unavailing. The possible presence of cardio-respiratory embarrassment cannot be too strongly stressed, and we have found in air-raid casualties that early intervention to close an open pneumothorax has been one of the best resuscitation methods at our disposal.

The place of oxygen therapy as an anti-shock measure is well established, especially so if there is any respiratory embarrassment. Even without the latter it should be instituted where possible, preferably with a B.L.B. mask. If there is any embarrassment, it should be continued until this is relieved.

The absence of shock is no indication for allowing the patient freedom of movement. On the contrary, all patients should be kept recumbent at least until the extent of the injuries are ascertained and should be disturbed as little as possible. Often the patient's position or clothing is sealing a serious chest wall defect, and until conditions are favourable for the immediate control of such a possible defect the greatest care is necessary.

When an X-ray unit is available, radiographs should be taken. These can be taken with little disturbance to the patient by gentle lifting on the blanket on which he is lying to put the plate in position, and lateral X-rays can also be obtained without disturbance. Lack of plant or the exigencies of the situation, however, may make X-rays impossible.

The chief function of the forward unit will be to relieve any embarrassment of the patient's cardio-respiratory mechanism, and secondarily to institute treatment which will decrease the incidence of complications.

The commonest causes of interference with normal cardio-respiratory function are: (1) Open pneumothorax, (2) tension pneumothorax, and

(3) much more rarely tamponade of the heart by blood in the pericardium. These three conditions will demand adequate treatment.

Open pneumothorax most commonly presents as a sucking wound and constitutes a serious menace to the patient, especially so if the defect is large. Its character makes recognition easy, and when recognised its control is equally simple. Temporary closure of the defect with a pad promptly relieves the patient's distress, and when this is done a fair assessment of his general condition can be made.

Treatment will depend on the time that has elapsed since receipt of the injury. If seen within the first eight hours, in carefully selected cases perhaps longer, and if the patient's general condition will allow, surgical revision of the wound should be undertaken. This entails excision of lacerated skin edges, torn muscle, the skin excision being enlarged if necessary to give full access to the deeper lacerated area, resection of broken rib ends and removal of loose splinters of bone, and excision of ragged pleural edges. Enlargement of the wound should be carried out to give adequate access to each of these layers if necessary. Where possible, blood should be aspirated from the pleural cavity and the wound closed by approximation of the muscles with interrupted sutures. When the defect is larger, some mobilisation of the muscle layer may be necessary. The skin can also be closed with interrupted sutures, but if there is any doubt, as, for example, in the longer standing cases, it should be left unsutured.

Temporary pleural drainage ideally should be carried out in all cases after revision by introducing a tube intercostally through a separate stab incision before the defect is closed. The chest should never be drained through the original wound. When, however, there is a possibility that the patient will be evacuated within forty-eight hours, tube drainage should not be instituted, as drainage entails attaching the tube to a water-seal system, and this would not be practicable during transport. At the end of forty-eight hours the tube is removed and a previously inserted suture tied.

Those cases without tube drainage will need aspiration at intervals to remove fluid and blood and encourage re-expansion of the lung, and even after tube drainage fluid collection will need aspiration until the pleura is dry and the lung is fully expanded. All cases without tube drainage should be clearly marked, preferably on the forehead, that aspiration is necessary. Failure to aspirate not only delays re-expansion of the lung, but continued presence of blood predisposes to infection. If the latter occurs, rupture of the wound is liable to occur, perpetuating the condition of open pneumothorax, and under much less favourable conditions.

One type of open pneumothorax is liable to be overlooked—that is, the valvular type, which prevents air entering freely into the chest, but allows of escape of blood. This type, however, has a characteristic feature, and that is the free escape of blood during expiration with cessation during inspiration. Respiratory embarrassment is not as marked in this type, but its recognition is none the less important. This group should be treated similarly to the sucking wounds.

Many of these cases will arrive too long after the injury to allow of revision, and should then be treated conservatively. The skin should be cleansed and

a pad of vaselined gauze or a pad of thick gauze wrung out in some mild antiseptic, covered if possible with oil silk, should be firmly strapped over the wound to make an air-tight cover. The wound should never be plugged. This is even now quite a common practice, which cannot be too severely condemned. If a pad only is applied, often in forty-eight to seventy-two hours the œdema and swelling of the muscles have effectively closed the gap in the chest wall and the wound no longer sucks; plugging the wound prevents this natural closure, and also greatly delays eventual closure.

Temporary suture of the skin may be justifiable as an extreme measure, but is only justifiable when the defect is large and sufficient material is not available to make an efficient pad and immediate transfer of the patient is necessary. If temporary suture is undertaken, this fact should be clearly labelled on the patient, preferably on his forehead, so that the state of affairs can be rectified at the earliest possible moment.*

It will be noted that no mention has been made of foreign bodies or of lung damage, and designedly so. Foreign bodies should not be searched for under these circumstances, but if seen during a surgical revision, they will of course be removed. Similarly, it is considered inadvisable that there should be any attempt in the forward units to deal with the damaged lung. This type of intervention needs the full armamentarium of thoracic surgery, which cannot be available under the conditions visualised.

Tension pneumothorax is the other condition which menaces life in the early hours of injury. The physical signs are absent breath sounds over a resonant or hyper-resonant hemithorax, displacement of the mediastinum, as shown by displacement of the apex beat, and of the trachea as felt in the suprasternal notch. These signs are accompanied by dyspnœa of varying severity.

Tension pneumothorax occurs most commonly without an external wound in association with crush injuries, but is not uncommon with penetrating wounds also. The condition results from injury to the lung, there being a valvular mechanism at the site of the injury which mainly allows of escape of air into the pleural space. The degree of tension will depend on the efficiency of the check valve, and this efficiency, together with the size of the opening, will determine the rapidity of its onset.

The aim of treatment is to relieve the tension within the chest by removal of air until there is no further embarrassment to respiration, and to maintain this state of affairs until the fistula is healed and further increase in tension cannot occur.

Symptoms may be severe enough to demand urgent intervention, and the simplest way to deal with the emergency is to introduce an ordinary serum needle through the second intercostal space anteriorly, when the air will be

* During the discussion on this paper, Col. Elliot Cutler, U.S. Army Medical Service, reverting to the question of first-aid treatment of chest injuries in the field, pointed out again that the loss of life from open pneumothorax occurred between the time of receipt of the injury and the patient's arrival at the forward surgical unit. He stated that he had given instructions to his regimental medical officers that when a sucking wound is present the skin should be sutured immediately and on the spot, so as to close the defect efficiently and enable the patient to be transported back to the forward unit with increased safety. This suggestion seems to be very sound and would help to save many lives which otherwise would be lost. It is a life-saving measure, and apparent transgression of surgical principles would appear to be thoroughly justified.

heard to hiss out of the needle. If there is not such great urgency, then the needle should be fitted with a long piece of rubber tubing, which should be led under the surface of some mild antiseptic, through which the emerging air will bubble. Cessation of bubbling will indicate that the tension has been relieved.

Early evacuation of the patient or recurrence of tension will demand retention of the needle *in situ*. The needle can be fixed firmly if it has been pushed through a disc of cork before being sterilised, and this can then be securely strapped to the chest. The needle should be attached to a water-seal drain if this is feasible; otherwise, a piece of sterile wool which is again covered by a layer of gauze should be strapped over the end of the needle to act as a dust filter. Its presence should be clearly indicated on the patient's forehead, so that it should not be overlooked for an unnecessarily long time at the next stage of evacuation.

The second interspace anteriorly is the best site for this type of case, as the lung is most commonly free at this site, and the needle will be less likely to be interfered with during transit by movement of the patient's arms.

Surgical Emphysema.—This condition is of common occurrence in chest injuries, more commonly with crush injuries than with penetrating wounds. Usually of limited extent, it then has little clinical importance. Rarely, however, the degree of emphysema may be severe and will demand some intervention to prevent its progress. In my opinion, the severe type is most often the result of an underlying tension pneumothorax, although it can occur when the area of lung damaged is attached to the chest wall. Quite obviously no good purpose is served by the older methods of multiple incisions—insertion of cannulæ with attempts to massage the air out through these exits. The underlying cause, being recognised, should be attacked. The tension pneumothorax should be relieved. It will be difficult to ascertain its presence clinically, and the only method of obtaining evidence may be blind needling of the pleura. This is preferable to leaving the condition unrelieved. If a tension pneumothorax is not present, then wide revision of the wound should be carried down to the adherent lung and the wound should be very lightly packed; firm packing will only perpetuate the condition. If there be two wounds, both may need revision.

Stove-in Chest.—This may constitute a serious embarrassment to the patient from the paradoxical movement of the chest wall. These cases result from crush injuries, and are commonly associated with a hæmothorax and acute dilatation of the stomach.

The paradoxical movement of the chest is sometimes quite obvious visually, and can be felt if not seen. Its control is necessary not only for relief of pain, but also to decrease cardio-respiratory embarrassment, the latter being produced in exactly the same way as with an open pneumothorax. Strapping the hemithorax often does not do this effectively, and a cardboard splint, if available, firmly strapped over the defect will suitably reinforce it. Strapping can also be reinforced by painting the outer surface with a paste of plaster of Paris.

It is necessary to cut a window in the strapping when a hæmothorax is

present to allow space for aspiration, the window of course being cut after the strapping is in place. Ordinary strapping is preferable to elastoplast.

In bilateral crushes the chest is encircled with strapping and preparation made for aspirating both sides if indicated. Morphia is necessary to ease pain and so allow secretions to be coughed up freely. In certain cases it may be urgently necessary to remove the secretions by intratracheal suction. If acute dilatation of the stomach occurs, the stomach will need decompression with a duodenal tube.

Hæmopericardium is not a common occurrence, and it is doubtful whether, even when diagnosed, anything should be done in the forward unit. American experience in civilian stab wounds among the negro population suggests that a large percentage of these cases can be treated conservatively, and only severe degrees of cardiac decompensation call for radical intervention. Even these will temporarily improve when blood is aspirated from the pericardium. These patients have a mounting pulse rate with a falling blood pressure (the latter occurring rapidly when compression is at all severe), enlargement of the area of cardiac dullness, and muffling of the heart sounds. The latter is also an index of severe bleeding, for except when the pericardium is fully distended the heart is pressed forwards against the anterior pericardial wall and the sounds may be clear.

Surgical intervention entails opening the pericardium and suturing the cardiac wound.

The commonest condition met with which does not menace life in the early hours is a hæmo- or hæmopneumothorax with or without a retained foreign body.

Hæmothorax will most commonly complicate the cases which have already been discussed, and is certainly the commonest complication of chest injuries. It may be simple in that there is little underlying damage, as with non-penetrating wounds and through-and-through gunshot wounds or wounds produced by small missiles without injury to the bony cage. Or it may be complicated where there is severe lung damage, as by a ragged shell fragment, or when one of the ribs has been fragmented and driven into the lung, this despite a relatively small superficial wound.

Under ideal conditions the aim and object of treatment should be the removal of every medium of infection—that is, foreign bodies of all kinds, metal or otherwise; removal of all damaged tissue and rapid re-expansion of the lung.

This entails a major thoracic intervention for properly selected cases; but in the forward area this is not advisable. This must necessarily mean that a certain percentage of these cases will develop late sepsis, either pleural or pulmonary, or both, and will need treatment at a thoracic centre. Many of them, however, will recover without complication, especially those with minimal lung damage.

Two groups of patients only should be considered for operative treatment. First, those in which it can be seen radiologically that the rib or ribs have been shattered on the entry side. These cases should be treated as though they were open pneumothoraces, and the wound excised and properly revised on the principles already laid down. Rib fragments should be removed from inside the chest, for in my opinion these cases inevitably lead to trouble, much

more so than the retained foreign body, and probably because the wounding agent in them is mainly rib and organic in nature.

Secondly, parasternal wounds should be revised, as in these the costal cartilage is often involved. These cartilages are notoriously susceptible to infection with a prolonged period of morbidity when not recognised.

Otherwise, these cases should be treated conservatively. The hæmothorax should be aspirated without gas replacement. The aspiration can be started twenty-four hours after the receipt of the wound and repeated daily or every other day, as much fluid being removed as possible at each sitting and a wide-bored needle used. The value of gas replacement is still a matter for debate, but the general attitude today is to deprecate its use. The advantages claimed for it are: (1) It makes aspiration easier. This cannot be denied, for by the introduction of air the negative intrapleural pressure induced by the aspiration is neutralised and makes further aspiration simple. (2) That by maintaining the pulmonary collapse fresh bleeding from the lung is prevented. This assumes that bleeding will occur after ordinary aspiration and is not supported by experience. (3) It is suggested that the introduction of air will aid the recognition of recurrent bleeding, which, if it occurs, is an index that the bleeding comes from the chest wall. Of this I have no definite experience, and it seems to me that recurrent bleeding would be equally well recognised if air is not introduced.

It is suggested that when the hæmothorax has been completely emptied and replaced by air, the air will be rapidly absorbed and the lung re-expand. This is quite contrary to experience; further fluid is poured out and a hydro-pneumothorax results which will need further aspiration.

The great objection to gas replacement is that it keeps the whole of the original pleural space involved, and if infection supervenes a pyopneumothorax results. Whereas, with aspiration without gas replacement, the pocket becomes smaller with aspiration and a smaller residual pocket is left if an empyema develops.

Hæmothorax patients usually run initial pyrexia, which is no index of infection. The temperature will settle in the course of a few days. Any recrudescence should be viewed with suspicion, and smears should be examined for organisms and where possible cultures should be obtained as a check. Infected hæmothorax is unlikely to occur during the early hours, except possibly as an anaerobic infection. Our experience with anaerobic pleural infections has been that they have not been extraordinarily fulminating, as with gas infections in general. Certainly the discovery of an anaerobic infection is no indication for immediate thoracotomy. Aspiration should be carried out in these cases as indicated, with the exception that the incision should be made down to the intercostal space, as has been advised by Tudor Edwards in putrid empyemata. The incision is packed and the granulation tissue forms a barrier to a cellulitic extension. Aspiration may now be carried out with impunity.

Recognition of the onset of infection will depend on the clinical state of the patient and the character of the aspirated fluid. The fluid has a purplish sheen and often an offensive odour. Smears and anaerobic culture will confirm the diagnosis if these are available.

Thoracotomy may be indicated as a life-saving measure in fulminating cases, and although I have no personal experience of such a case, I should be inclined to do a wide thoracotomy as advised by Neuhof for putrid empyema and lightly pack the pleural cavity with gauze impregnated with zinc peroxide emulsion.

It is of great value in any infected case undergoing aspiration to keep a portion of fluid from each aspiration in a tube. This gives an indication of the rate at which pus is forming, and after standing twenty-four hours gives an index of the amount of pus. In ordinary cases drainage will not be indicated until at least 75 per cent. of pus is present.

Abdomino-thoracic injuries form a very serious group of injuries. Gordon Taylor states that they should be left to the end if there is such a pressure of work on other less seriously injured cases as to leave little or no time.

The serious element in these cases is the abdominal part, and wounds involving the left cupola are more serious than those involving the right. On the left side the stomach and splenic flexure are liable to injury, whereas on the right the missile tends to lodge in the liver. The upper abdominal structures are most commonly damaged, but the lower abdomen may also suffer if the direction of the missile is more vertical. For practical purposes the chest injury can be treated conservatively, or at least on its merits, at this stage while the abdominal injury is assessed and treated. Should the chest need operative intervention, as with a sucking wound, the diaphragmatic wound can be excised and enlarged and the upper abdomen explored and visceral injuries dealt with, the diaphragmatic incision then being closed after pinching the phrenic nerve.

If no thoracic intervention is indicated, laparotomy is the procedure of choice.

Chemotherapy.—The use of drugs of the sulphonamide group is so universal at the moment that it ill becomes anyone to raise even a mildly dissentient voice. Their value in the main is proven, but at the present it would be unwise to feel that we yet have the complete answer. There is a tendency to depend too much on chemotherapeutic aids, and not infrequently one has seen cases where basic surgical principles have been violated under the cloak of chemical administration and application. A critical attitude to chemical preparations should still be maintained, and no whit of surgical technique should be omitted through a possibly misguided faith in their efficacy. We should at present continue the use of this group of drugs both prophylactically and therapeutically, internally and by superficial application, but clinical records should be kept to guide those who will later be responsible for the care of the patients. External application should be reserved for those who are being treated conservatively. There surely can be no warranty for covering a surgically revised wound with the powdered drug, and in fact personal observation points to inhibition rather than acceleration of smooth healing.

SMALLPOX VACCINATION AND PULMONARY TUBERCULOSIS

By R. Y. KEERS and P. STEEN.
From Tor-na-Dee Sanatorium, Aberdeenshire.

A search of the literature gives little information regarding the possibility of vaccination for smallpox being the causative factor in a subsequent flare-up of latent or active pulmonary tuberculosis. Blacher (1931) has recorded two cases, both in children. In the first of these a boy aged 11, suffering from dystrophia adiposo-genitalis, developed a tuberculous meningitis following re-vaccination, and from this Blacher concluded that the vaccination had re-activated a pre-existing tuberculous focus. His second case was that of a girl aged 11, whose skiagram showed a small hard focus in the right upper zone. She was subsequently vaccinated, and ten days later there was fever and X-ray evidence of re-activation of the pulmonary lesion.

Ainger (1937) recorded two further cases where tuberculous meningitis followed immediately on vaccination, and from this he drew the conclusion that either vaccination lowered the powers of resistance, thus paving the way for a fresh infection, or that an inactive lesion already present flared up as a result of the procedure and spread unopposed throughout the lung.

Stone (1931) reported the results following the vaccination of 337 patients at the Robert Koch Hospital, St. Louis. All stages and types of pulmonary tuberculosis were included in Stone's cases, and only one patient showed any definite pulmonary exacerbation, while two others had a temporary increase in the amount of cough and sputum. His view, therefore, was that the presence of pulmonary tuberculosis was not a contra-indication to vaccination.

In the summer of 1942 there was an outbreak of smallpox in Glasgow, and later in the same year in Edinburgh and Fife. Considerable numbers of the public were vaccinated, and one of us (R. Y. K.) received numerous requests from former patients of the sanatorium for advice as to whether, in view of their previous pulmonary infection, they should undergo vaccination. Those living or working in Glasgow were advised without hesitation to be vaccinated, as it was felt that the results of smallpox would be much more disastrous than any post-vaccinal flare-up in the chest. As far as is known, none of those so advised suffered any ill-effects. Later in the year four cases were admitted to the sanatorium, all of whom gave a history of vaccination followed almost immediately by the appearance of symptoms of pulmonary tuberculosis.

Case Records

Case 1.—Male, aged 28. This man, an engineer by profession, had an excellent medical history and for years had not been off work for a single day. In June, 1942, he applied for a post abroad, and before acceptance he underwent and passed a medical examination. A condition of his appointment was that he must be vaccinated in this country before departure, and this vaccination was duly carried out by his own doctor in July. Four days following the

vaccination he had a severe reaction; he felt feverish and his arm was swollen and tender. After a further three days he developed a sharp pain in the left chest, which proved to be the beginning of an acute pleurisy with effusion. The subsequent skiagram revealed bilateral infiltration with cavitation in the left upper zone. This patient stated most emphatically that, prior to vaccination, he had felt perfectly well and had been able to do his work, which entailed considerable physical effort, without the slightest inconvenience.

Case 2.—Male, aged 22. This boy gave a history of pulmonary tuberculosis dating from the age of 16, for which he had received sanatorium treatment on several previous occasions, the last being in 1939. Following this he had remained fairly well and had been living quietly at his home for two years, where his main occupation had been fishing. In July, 1942, he was vaccinated and had a severe local reaction with, at the same time, pain in the chest and dyspnœa. Radiological examination a few days later showed the presence of a small pleural effusion on the right side together with a fresh area of exudative disease in the mid and lower zones.

Case 3.—Male, aged 20. This boy had been treated in the sanatorium in 1941 for a left pleural effusion, from which he made a completely satisfactory recovery. He was discharged after a six months' stay and spent the spring and summer of 1942 as junior master in a preparatory school. In the autumn he was in business in Edinburgh, still well and free from symptoms. In November, 1942, he was vaccinated. He had very little local reaction but felt generally "ill," his main symptom being lassitude. He did not feel well enough to return to business, and three weeks later, in addition to the lassitude, he developed a slight temperature associated with the appearance of cough and sputum. Tubercle bacilli were present in the latter, and subsequent X-ray examination showed the presence of a recent area of exudative disease in the right upper zone.

Case 4.—Female, aged 19. This girl was working in an emergency hospital as a V.A.D. and was vaccinated along with her colleagues in July, 1942. She had a severe local reaction and was in bed for four days. Subsequently she felt tired, and three weeks later had the misfortune to fall victim to a mild epidemic of glandular fever which attacked some of the hospital staff. She recovered rapidly from the fever but the lassitude previously present persisted, and shortly after she had a sudden hæmoptysis. Radiological examination showed scattered infiltration throughout the left upper and mid zones, with commencing cavitation immediately below the clavicle.

Discussion and Summary

In view of the relatively few references to the association between vaccination and pulmonary tuberculosis which we have been able to find it is felt that these cases should be recorded. It is impossible to draw any definite conclusions from isolated instances such as these, but it would appear that there is sufficient evidence here to justify the assumption that vaccination may cause a flare-up in a latent focus.

Our results are at variance with those reported by Stone, but it should be remembered that his cases were under sanatorium conditions at the time of vaccination, while those we have recorded were engaged in their normal occupations, and therefore no more precautions were taken in their cases than would be taken with the average healthy individual.

The necessity for widespread vaccination of the population will not, we hope, arise again, but should it so happen it would be well to exercise special caution before submitting to vaccination known cases of pulmonary tuberculosis.

REFERENCES

AINGER, A.: *Wein. Med. Wschr.*, 1937, **87**, 441.
BLACHER, W.: *Jb. Kinderheilk*, 1931, **130**, 201.
STONE, R. E.: *Amer. Rev. Tuberc.*, 1931, **23**, 706.

BRONCHOGENIC NEOPLASM IN A BOY OF TEN

BY RONALD JONES, K. W. MACKENZIE AND E. BIDDLE.
From the East Suffolk and Ipswich Hospital.

THIS case presents unusual and interesting features, and it has been thought worth while to record it because bronchial neoplasm occurring in one so young is rare and also because asthma as a presenting symptom of bronchial growth is unusual. It is not given by standard textbooks or by Davidson,[1] and leads, as in this case, to diagnostic difficulties and delay.

A.B., aged thirteen, was admitted to hospital on May 12, 1936, with two weeks' history of bronchitis. There had been no dyspnœa or chest pain. During the week preceding admission he had vomited once or twice daily. Past history: winter cough and inoffensive, non-purulent sputum, about half a cupful in each twenty-four hours, since the age of ten years. No hæmoptysis. No pneumonia, pleurisy or other chest illness. Whooping-cough at the age of six years and mumps were the only exanthemata reported. Family history: parents well, two sibs well.

On examination he was well nourished, weighed 5 st. 9 lbs. Throat healthy. The heart was not displaced; the lungs showed signs of bilateral basal bronchitis only. There was no clubbing of the fingers. Abdominal examination was negative. The sputum was examined for tubercle bacilli on two occasions with negative results. An X-ray on May 25, 1936, showed a raised, immobile right diaphragm. The appearances were those of right basal atelectasis, and bronchiectasis was considered likely. Bronchography was carried out on June 3, 1936, with inconclusive findings. During these eighteen days in hospital the temperature was normal except on two occasions, when it was 99°. Considerable dry cough was present throughout. The child was then taken home by his parents and the matter was not further pursued. The weight on discharge (June 8, 1936) was 5 st. 9 lbs.

He was next seen in out-patients on January 6, 1938. On examination there was definite dullness and crepitus in the right lower zone and the patient coughed up some thick, yellow, slightly offensive sputum.

He was re-admitted on March 16, 1938, on account of further attacks of asthma. Since his previous admission he had had measles. On examination his general condition was good and his weight was 6 st. 13 lbs. Throat healthy. C.V.S.: no cardiac enlargement or displacement, sounds normal, regular

rhythm. Chest: no clubbing of fingers, trachea central, slight dullness and crepitus in the right lower zone. Vocal resonance normal. Abdomen: nil, abnormal. Nervous system: nil, abnormal. Bronchography was carried out on March 18, 1938. The left bronchus and its branches were well outlined and normal. On the right side, the upper lobe bronchi filled well. Bronchial block was present and prevented filling of the right mid-lobe. There was partial filling with lipiodol of the bronchi to the lower lobe, and these were bronchiectatic.

On April 8, 1938, under general anæsthesia, bronchoscopy was performed. The left bronchus was normal in appearance and movement. The right main bronchus was completely obstructed distal to the eparterial branch. The appearance was suggestive of granulation tissue, and probing failed to reveal any foreign body. The tissue was soft and bled easily, but some was removed, despite difficulty with hæmorrhage.

On April 27, 1938, at bronchoscopy, a further attempt at clearing the granulations was made, but pathological examination failed to reveal the presence of new growth. By May 7 there was a considerable reduction in the quantity of sputum. On this date, and subsequently on the 13th, further tissue was removed, leaving a channel through which the middle and lower lobe bronchi could be seen. A small portion of this growth removed ante-mortem per bronchoscope was examined microscopically. It showed columns of deeply staining cells in a fibrous stroma containing many inflammatory cells and small areas of necrosis. It looked somewhat like a basal-celled carcinoma, but a few of the deeper columns suggested a tubular structure (Fig. 1).

During a further bronchoscopy on May 20, 1938, the patient died. On the postero-lateral wall of the right main bronchus was an area ¾ by ½ inch in the position already indicated, which was hard to the feel of a probe.

At autopsy on May 20, 1938, a papillary growth was seen extending into the lumen of the main bronchus of the right lung, distal to the origin of the right upper lobe bronchus (Figs. 2 and 3). This bronchus was almost completely occluded, thus causing bronchiectasis of the right lower and middle lobes. There were enlarged hilar and tracheo-bronchial glands, sections of which showed no malignant cells. No secondary deposits were found in the chest, abdomen or skull.

The growth removed at autopsy was a sessile tumour, 1·5 by 1·7 cm. and 0·9 cm. thick. The surface was smooth and ulcerated. On section it was uniformly greyish-white in colour and devoid of necrotic areas. It was a well-defined tumour. Microscopically the superficial areas resembled the portion removed ante-mortem, but in the deeper area the tumour tissue was seen to be infiltrating the bronchial wall. Tubular formation here was much more evident, and neither duplication of the epithelium nor rupture of the basement membrane was seen. The cells were fairly large, with scanty granular cytoplasm and a large and well-defined nucleus of uniform size, which failed to show any marked signs of activity, but the nuclei rather varied in the density of their staining (Fig. 4).

At the time of writing this report (1936) little was to be found in the literature with reference to benign epithelial neoplasms of the bronchi, and this tumour was described as a well-differentiated carcinoma.

References in the literature to bronchial neoplasms in young people are rare. They are often unsatisfactory in that the origin of the growth from the

PLATE XXI.

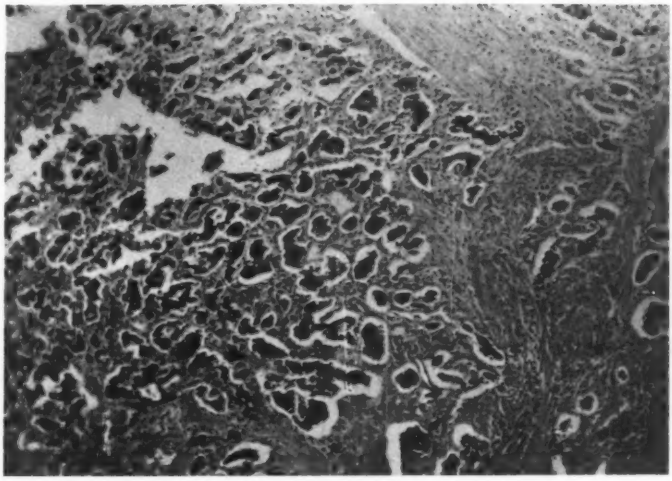

FIG. 1.—SUPERFICIAL AREA OF TUMOUR. ×35.

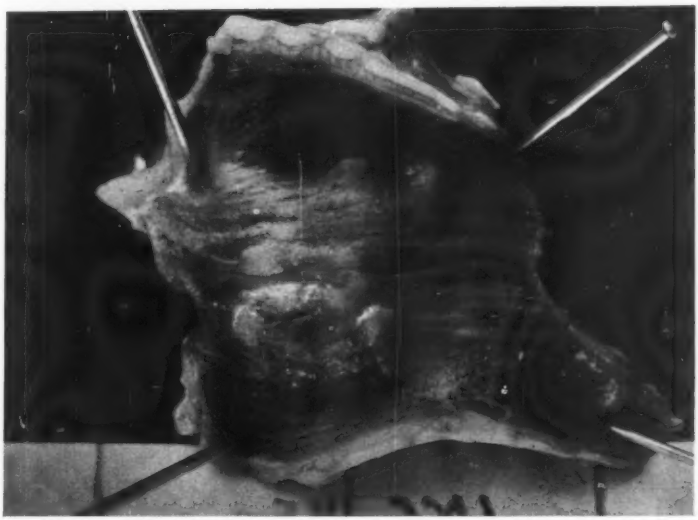

FIG. 2.—BRONCHIAL TUMOUR. ×1½.

[*To face page* 114.

PLATE XXII.

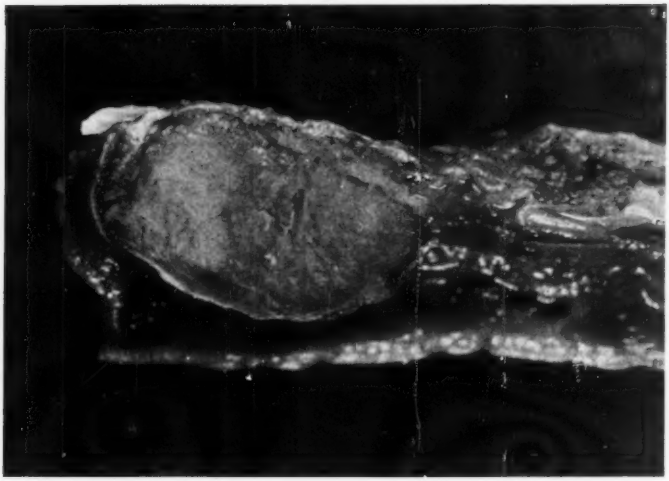

FIG. 3.—SECTION OF BRONCHIAL TUMOUR. ×3.

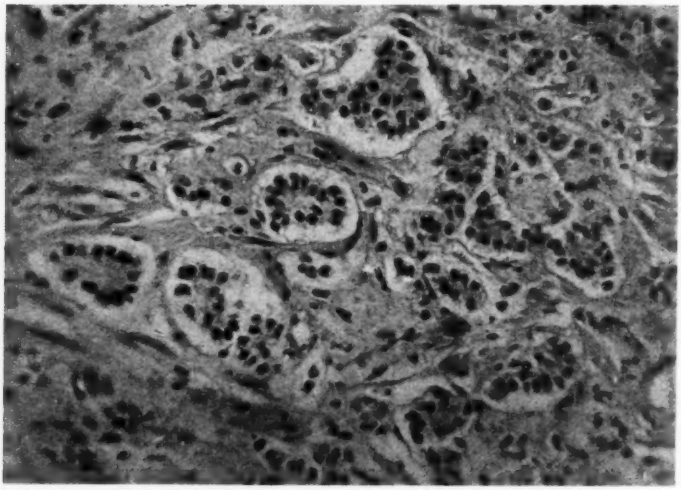

FIG. 4.—DEEPER PORTION OF TUMOUR. ×130.

[*To face page* 115.

bronchus is not always evident and in that bronchial adenoma was not always differentiated—as in this case—from carcinoma. Maurice Davidson,[1] reviewing a series of 107 cases of primary lung cancer at the Brompton Hospital, found 2 under 21, one of these being a female aged 16½ (no histological record) and the second case a male child of 5 years in which sarcoma of the thymus was present. Duguid's figures from Manchester Royal Infirmary, quoted by Davidson,[1] gave 5 out of 173 cases under 21. Sommer[2] reports a case of a girl of 7 years who died after 7 weeks' illness. At post-mortem a rounded, tense, encapsulated mass was found in the right upper chest, 4-5 inches in diameter. This was probably a carcinomatous degeneration of a congenital cyst rather than a primary neoplasm. Beardsley[3] records a bronchial adeno-carcinoma in a female infant aged 10 months who died after 6 months' illness. Fried,[4] analysing 47 cases, found only 1 case of bronchial carcinoma between 20 and 29.

The case most resembling the present one is described by Wasch, Lederer and Epstein,[5] who diagnosed an adeno-carcinoma of the right upper bronchus by bronchoscopy in a boy of 11 years of age. He died 7 years later, during which period he developed an eosinophilic adenoma of the pituitary gland and gigantism. This was undoubtedly a bronchial adenoma. Foster-Carter,[6] in a personal study of 22 cases of bronchial adenoma, found the average age at the onset of symptoms 28 years, the youngest being 21 years. In a study of 47 published cases of bronchial adenoma, he found 4 patients aged 10-14 years at the onset of their symptoms. He mentions asthma as a frequent symptom.

The history of the present patient suggests that he had bronchiectasis, presumably the result of bronchial obstruction, by the age of 10, so that the total course of this illness was therefore at least three years. His good general condition and the increase in body weight between the first and second admissions to hospital are noteworthy.

Bronchial adenomata, which number 5-6 per cent. of all bronchial neoplasms, are less rare in early life than carcinomata. However, even typical adenomata may show malignant features—even metastasis to liver[7]—and therefore an histological report of carcinoma is liable to be given. A long history and especially youth of the patient should, as in this case, lead to closer consideration of adenoma *v.* carcinoma as the diagnosis.

We are indebted to Dr. Foster-Carter for his histological assistance.

REFERENCES

ANDERSON, W. M.: *Journ. Thorac. Surg.*, 1933, **12**, 351.
BEARDSLEY, J. M.: *Canad. Med. Ass. Journ.*, 1933, **29**, 257.
DAVIDSON, M.: Cancer of the Lung, 1930, Wright, Bristol.
FOSTER-CARTER, A. F.: *Quart. Journ. Med.*, 1941, **10**, 139.
FRIED, B. M.: *Medicine*, 1931, **10**, 373.
SOMMER, A. W.: *Min. Med.*, July, 1934, **17**, 415.
WASCH, M. G., LEDERER, M., and EPSTEIN, B. S.: *Journ. Pediat.*, October, 1940, **17**, 521.

PNEUMOPERITONEUM COMBINED WITH ARTIFICIAL PNEUMOTHORAX AND PHRENIC PARALYSIS

By R. Y. KEERS.

From Tor-na-Dee Sanatorium, Aberdeenshire.

PNEUMOPERITONEUM is gradually finding a place in the collapse therapy of pulmonary tuberculosis, and encouraging accounts have appeared recently in the American literature (Rilance and Warring, 1942; Fowler, 1942).

The following case is put on record because it shows the value of a combination of methods and also because of the very satisfactory diaphragmatic elevation produced by a pneumoperitoneum at a time when the patient was going downhill.

Miss B., aged 31, was admitted to Tor-na-Dee Sanatorium on January 8, 1942, with a history of a succession of chest "colds" throughout the preceding four months, culminating in December in a sharp hæmoptysis. She was toxic, the evening temperature reaching 101°, sputum contained tubercle bacilli, and the sedimentation rate gave a reading of 46 mm. in the first hour. The skiagram (Fig. 1) showed infiltration involving all zones of the right lung, with cavitation in the mid zone, and there was also a small area of infiltration in the left mid zone. A right artificial pneumothorax was induced immediately and was quickly followed by a reduction in the fever. The sputum, however, remained unaltered in amount, and subsequent X-ray examination, while indicating a fairly good collapse with an apparent absence of adhesions, showed that the mid-zone cavity was still patent. The pneumothorax was accordingly supplemented by a right phrenic evulsion, but in spite of this the cavity remained unaltered (Fig. 2). A thoracoscopic examination was carried out on August 8, 1942, and the cause of the difficulty was revealed in the form of two short, thick adhesion bands lying posteriorly, immediately over the site of the cavity. Unfortunately both these bands contained lung tissue and no attempt could be made to divide them. A further thoracoscopy was done two months later, but the appearances inside the chest were unaltered, and any thought of pneumolysis was finally abandoned. By this time the patient had begun to go downhill: continual mild evening pyrexia persisted, she was losing weight slowly but steadily each month, and in September there was evidence of extension of the lesion in the left mid zone. The question of an extensive extra-pleural pneumothorax was considered but abandoned, and finally, on the suggestion of Dr. F. H. Young, to whom the skiagrams had been submitted, it was decided to induce a pneumoperitoneum. The induction was carried out on February 23, 1943, with the introduction of 800 c.c. of air. Refills have been given since at weekly intervals, and Fig. 3 shows the appearance on May 6, 1943. At this time the total elevation of the diaphragm was 12 centimetres. A small pleural effusion appeared in the pneumothorax space in the beginning of March, but this was not associated with any constitutional upset, and only small refills of the A.P. at intervals of two weeks have been necessary.

Improvement in the patient's condition has been dramatic. The tem-

PLATE XXIII.

FIG. 1.

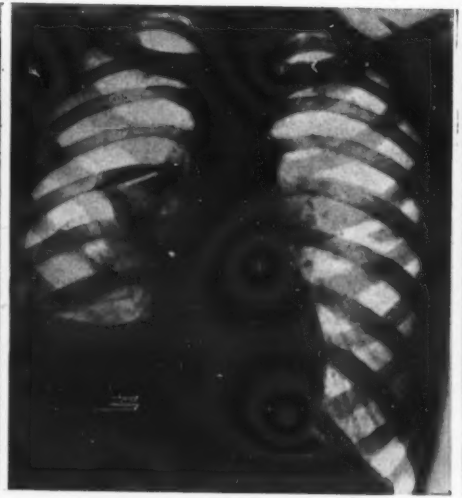

FIG. 2.

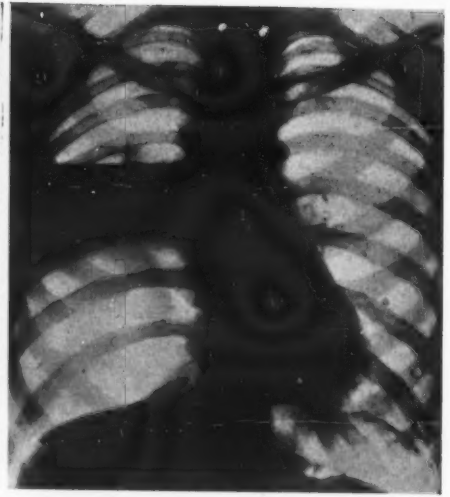

FIG. 3.

[*To face page* 116.

perature rapidly settled, cough and sputum diminished steadily, and the latter became T.B. negative, both on direct examination and after concentration, within eight weeks. In addition, during the seven weeks following the induction of the pneumoperitoneum the patient gained 8½ lbs. in weight compared with an average monthly loss of 2 lbs. since the previous May.

It is, of course, much too early to forecast the outcome of this case, but the procedure has already been justified by its immediate effects. There would appear to be a place for pneumoperitoneum, used in conjunction with phrenic paralysis, in the treatment of basal and mid-zone lesions.

REFERENCES

RILANCE, A. B., and WARRING, F. C.: *Amer. Rev. Tuberc.*, 1941, xliv., 323.
FOWLER, W. O.: *Amer. Rev. Tuberc.*, 1941, xliv., 474.

POLYSEROSITIS

BY HANNES HANNESSON.
From the County Sanatorium, Lenham, Kent.

Introduction

THE generic term " Polyserositis " includes a well-defined group of cases with chronic inflammatory thickening of the membranes lining the great serous cavities. Often there are recurrent effusions, and the chief symptoms may be those of heart failure. Many of these cases are regarded as having a tuberculous origin in all probability, though proof of their exact nature may be difficult to produce during life. Chronic tuberculosis of the great serous sacs leading to polyserositis is regarded as a condition associated with a high degree of immunity to the infection; in the long run the clinical picture becomes slowly transformed as the infective element is submerged in mechanical obstruction to the heart and circulation.

Polyserositis is much more frequent in its incidence than was formerly believed; and it has a wide variation in clinical symptomatology and in pathological findings. The nomenclature is involved and confusing though polyserositis is probably the most satisfactory term. Several conditions clinically similar are included under this title: Concato's disease and Pick's disease indicate syndromes with characteristic features.

In the last ten years I have met a number of cases in which there was a chronic hyperplastic serositis of the pleural, peritoneal and pericardial cavities. This experience and a study of the literature form the background of this paper. Though there are some admirable papers on polyserositis scattered throughout the literature, there are few reviews, a state of affairs which led the writer to consider it worth while to attempt another.

Historical Note

Laennec (1819) first drew attention to the fact that adhesions followed the absorption of a pericardial effusion. The first reference to polyserositis was in 1842, when Rokitansky, the great Bohemian pathologist and anatomist,

described the condition in his "Handbook on Special Pathologic Anatomy." Van Deen (1846) wrote the first paper on its clinical aspects. Fagge (1875) and Wilks and Moxon (1875) later recorded excellent descriptions of the syndrome. Concato's original article appeared in 1881. The Italians refer to the condition as Concato's disease and in France the condition is known as perivisceritis. Curschmann (1884) studied the syndrome from the pathological aspect, naming it "Zuckergussleber" (sugar-iced-liver). Tissier (1885), Picchini (1891) and Osler (1896) have also reported cases.

Polyserositis did not attract much attention until Pick (1896, 1899, 1900) reported three cases of chronic obliterative pericarditis with ascites, adhesions and effusion in the pleura, fibrinous peritonitis, perihepatitis, cirrhosis and perisplenitis. Polyserositis and Pick's disease have often been confused in the past, since it is true that the latter condition may result from the former and the two conditions may co-exist. Cabot (1898), Herrick (1902), Nicholls (1902), Kelly (1903), Evans (1918), Reid (1920), Mayo (1922) and Dawd (1923) have all written on this aspect of the subject.

Ætiology and Pathogenesis

The essential cause of the syndrome is unknown, although recorded cases in the literature reveal certain common clinical and pathological features which suggest also a common ætiological factor.

Three hypotheses have been put forward in explanation:

1. That the condition is an infection by an organism of low virulence, capable of provoking marked fibrosis in a serous membrane in a patient with low resistance, inherent or acquired. A number of pathogenic organisms have been held responsible: Mycobacterium tuberculosis, Mycobacterium lepræ, Bacterium typhosus, Bacterium coli, Treponema pallidum, Plasmodium malariæ, Streptococcus pneumoniæ.

   Rheumatic fever, whooping cough and diseases of the gall-bladder and liver have from time to time also been held to be ætiological factors in some instances of polyserositis.
2. That the condition is due to a toxin of unknown origin and nature.
3. That the condition is a state of allergy.

The disease is equally common in both sexes and occurs at all ages, though more commonly in the second and fifth decades. It is not very rare. Reid (1920) discovered fifteen examples (0·4 per cent.) in a review of 3,900 necropsies at the Massachusetts General Hospital.

The syndrome is considered by many authors as probably toxic in origin, the source of the toxin being unknown. The wide distribution of lesions affecting several or all of the serous membranes supports this view. In many patients there is no history of any antecedent illness, while in others there is an acute onset in which one membrane only is involved; for example, the disease may follow a pleurisy or pericarditis. Occasionally acute infections such as rheumatic fever, syphilis, typhoid fever and malaria are followed by polyserositis. In others it is a terminal event in patients who have suffered from organic disease, and it is not always easy then to regard the latter as

necessarily the sole explanation of the polyserositis. For instance, it may be associated with arteriosclerosis, chronic interstitial nephritis and uræmia.

Many writers believe that polyserositis is caused by direct bacterial infection of the serous membranes. The evidence is far from convincing, though there is one group of cases in which a tuberculous ætiology can be regarded as certain, for the organisms can be isolated from the lesions by careful search. How large a portion of all the cases is formed by this group is not settled; recent work suggests that it is considerable.

Some authorities hold that the disease is chemical in nature and has its origin in the liver. They postulate an unidentified toxin passing from the liver to the peri-hepatic peritoneum and believe that the condition always begins as a peri-hepatitis.

Nicholls (1902) described a case in which he believed that the source of the inflammation was the gall-bladder. Erskine (1935) records a very interesting case of polyglandular dystrophy accompanied by polyserositis. Stoll (1929) reports a case of trichinosis simulating polyserositis, and Reed (1935) reports another where a clinical diagnosis of fungus infection and Pick's disease was evident. These isolated associations are almost certainly fortuitous. The diseases mentioned are encountered so often without polyserositis that it is hard to believe that they can ever play any part.

The only case reported in the literature of polyserositis due to Mycobacterium lepræ is recorded by Ying (1936). Acute rheumatic fever with its proclivity for causing effusions would seem a likely cause of polyserositis in certain instances. Such cases have been recorded (Matthew, 1912). Slight degrees of polyserositis are common in acute rheumatic fever in children. In some of these the toxic character of this rheumatic serositis has also been marked by considerable renal irritation as shown by albuminuria with cylindruria (Mosler, 1910).

Both Rosenbach (1907) and Osler (1926) have recorded polyserositis with constrictive pericarditis and rheumatic valvular lesions. This combination may be termed the " Rosenbach-Osler " syndrome, so separating it from Pick's disease, where constrictive pericarditis is found without rheumatic valvulitis and its consequent cardiac enlargement. A clear separation must be made also between true polyserositis of inflammatory origin and " pseudo-polyserositis " due to chronic effusions of purely mechanical origin; for example, chronic hydrothorax with ascites, where the cardiac background and type of effusion are distinctive.

The hypothesis that polyserositis is a state of allergy requires consideration. Boyd's contention that rheumatism manifests itself in allergic reactions of serous surfaces provides an explanation for one type of polyserositis. Mallory (1929) supports this view. There are many reports of polyserositis occurring during the course of rheumatic polyarthritis (Paul, 1930; Lenoble and Pineau, 1927). Chappelle (1933) found no evidence in his histological studies of the diaphragm, liver or spleen to indicate the presence of old or recent vascular or peri-vascular lesions. He inferred that in his cases he was dealing with the end stage of a process in which identifiable lesions may be found. Volhard (1923) apparently did not consider or recognise inflammation as an ætiological factor in so far as he held that the " zuckergussleber " was the result of the

chronic ascites rather than the cause. In this connection it is important to realise that in no known state of allergy is there a specific histological lesion.

Patterson (1917) in his experimental work with pleural effusions found that the pleural cavity of rabbits, inoculated with virulent tubercle bacilli after a pneumothorax had been established, did not respond to the inoculation with the development of fluid. He further discovered that a second inoculation of bacilli into the pleural cavity some weeks later resulted in a rapid accumulation of bloody serous exudate rich in leucocytes. The mechanism of the altered reaction to infection in animals already tuberculous may be explained by an allergic condition of the fixed tissue cells, this allergy being produced by an already existing focus of disease in some other part of the body.

Manwaring and Bronfenbrenner (1913) studied the intraperitoneal lysis of tubercle bacilli and concluded that this is not due entirely to substances in the circulating fluids but to specific changes in the fixed peritoneal cells of tuberculous animals. Much and his co-workers (1911) and Kraus and Hofer (1912) have obtained evidence of lysis of tubercle bacilli in the peritoneum of an infected animal. Rist, Kindberg and Holland (1914) produced an immediate bloody, ascitic effusion in tuberculous guinea-pigs by the intraperitoneal injection of living tubercle bacilli. Howard and de Veer (1936) have confirmed the findings of others that serous effusions may be elicited in several serous cavities of tuberculous guinea-pigs by the introduction of tuberculin into one of the cavities. Krause and Willis (1926) have reported the development of fibroid change in the pleura and peritoneum following the repeated subcutaneous and intracutaneous injection of living tubercle bacilli in guinea-pigs previously rendered allergic and more or less immune by inoculation with a strain of organisms of low virulence. Those who favour the humoral theory of immunity consider that the virulence of the injected bacilli is lessened by the action of the serum of the effusion in such a manner that the tissues would react differently to these attenuated bacilli than they would do to an implantation of unaltered virulent bacilli. This does not explain why there is an exudation in the tuberculous and not in the normal controls which is the primary and fundamental difference in the reactions.

Pathology

The inflammatory process may start either below or above the diaphragm, depending upon the concentration and virulence of the offending agents and the local resistance to the structures affected. Wherever the process begins the ultimate lesions are identical. De Renzie (1898) believed that the peritoneum is first affected, then the right pleura and then the pericardium; he found that if the right pleura was involved first, the disease usually extended to the peritoneum and thence to the left pleura and the pericardium.

The prominent lesions observed consist in a thick fibrous, whitish, glistening connective tissue encasement of the heart, liver, pleura, peritoneum, omentum, mesentery and intestines. The fibrosis may be greater on the right side of the body possibly because there are more lymphatic channels through the right than through the left cupola of the diaphragm.

The association of pericarditis with peri-hepatitis is the usual finding, and this condition develops into an adhesive pericarditis with gradual interference

with cardiac function and the development of the lesions associated with it. The condition generally pursues a remarkably slow and insidious course, giving rise to exudation of large quantities of sero-fibrinous fluid in the pleural and pericardial sacs. The fluid portion of the exudate is usually re-absorbed, leaving the characteristic fibrinous substance deposited upon the viscera involved, with ultimate development of dense adhesions to the surrounding tissue undergoing cicatricial contraction so that distortion of the involved viscera occurs. Smith (1913) found the incidence of adherent pericarditis to be 62 in a series of 3,052 necropsies.

A characteristic feature of this syndrome is that the serous covering of the liver is entirely free from the fibrosis so that the fibrous casing can be separated in layers, leaving the liver more or less intact. This finding distinguishes it from other forms of peri-hepatitis which involve not only the capsule of the liver but also the liver substance itself. The liver shows no other structural change apart from compression and distortion. Portal cirrhosis of the Laennec type is occasionally associated with polyserositis.

The pathological findings are fairly constant. In fully developed cases changes are found in all the serous surfaces, but the changes in the pericardium and around the liver are the most obvious and most constant. Dense adhesions, which may be calcified, are found in the pericardium. The heart is often enlarged, and valvular disease may be present. Adhesions are found in both pleural cavities, the right side usually more advanced than the left. There is often an enlarged spleen with perisplenitis. The process apparently starts as a low-grade chronic inflammatory reaction of the peritoneum lining the diaphragm and upper abdomen. The similarity of mediastino-pericarditis and polyserositis and the fact that the former may result from the latter makes it obvious that they are not easy to differentiate. It is possible that the two conditions are not essentially different.

Microscopically, the fibrous encasement consists of newly grown fibrous tissue arranged in layers with advanced hyaline degeneration (hyaloserositis of Nicholls, 1902, 1903). This fibrinous deposit is then invaded by leucocytes, mast cells and fibroblasts. There are very few bloodvessels and only a small amount of round cell infiltration. There is a tendency toward lamellar formation. This peculiar overgrowth of fibrous tissue may become hyalinised, giving the deposit a glistening appearance. It may even resemble a cartilaginous substance, and a deposit of lime salts may give rise to the formation of bone-like plaques (Stone, 1926; Ramsay, 1937). Rolleston (1912) states that microscopically the liver shows irregular areas of fibrosis, but generally the amount of fibrosis is scanty and may be absent in considerable areas. He describes a definite microscopic difference between the deposits on the serous surfaces, especially the liver, in Pick's disease and Concato's disease. In the former the deposit is under the capsule, whereas in the latter the fibrous overgrowth is outside the capsule.

The pericardial fluid removed by tapping may be a transudate due to venous stasis or an exudate. This fluid may be straw-coloured, amber, hæmorrhagic or purulent. The fibrin content is high and the cells are increased in number. Polymorphonuclear cells may predominate, but usually monocytes and lymphocytes are found in great numbers. If hæmorrhagic,

the fluid may resemble the fluid from rheumatic fever exudates or from those resulting from metastatic tumours in serous membranes. Sometimes the fibrinous exudate becomes very thick and pools of fluid, serum or pus are formed here and there—the so-called loculated pericarditis.

The fluid obtained from the pleural cavities is usually an exudate. It is clear or yellow, may be cell-free, sterile and contains about 3 per cent. of albumin. The specific gravity is 1·018 or higher.

The ascitic fluid is usually clear and straw-coloured with about 3 per cent. of albumin which consigns it to the class of inflammatory exudates. The specific gravity is 1·105 or higher. Endothelial and lymphocytic cells may be present. No organisms have been identified in the fluid.

The mode of development of the serositis is often difficult to determine. The published cases (Kelly collected only 39 cases from the literature up to 1903) show that the original inflammation may start in any serous membrane. In five of Kelly's cases, however, there was a distinct history of an antecedent pericarditis, in one as long as twenty-three years before the onset of the ascites. Matthew (1912) records a case in which the inflammation undoubtedly originated in the pericardium. Taylor (1900) thought that the peritoneum is attacked first, not the pericardium, but the evidence he offers is not convincing. The history may help a little in deciding the place of onset. When the condition has begun as a perihepatitis or as a peritonitis ascites occurs within a short time, perhaps a few weeks or at most a few months. There will be no œdema of the feet and legs. When the serositis begins in the pericardium the ascites does not appear early. There may be slight œdema of the ankles and some breathlessness due to the adherent pericardium causing cardiac embarrassment, and after a period of years the ascites appears and persists.

With regard to the cause of the ascites, it would appear that in the absence of extensive liver cirrhosis or much involvement of the portal vein at the hilum of the liver, the peritoneum itself must be held mainly responsible. Of secondary importance is the damaged myocardium, for in myocarditis relief is afforded to the impeded heart by the occurrence of ascites, and œdema of the extremities occurs only terminally. Of lesser import is the slight implication of the portal vein at the portal fissure, the resistance offered to the portal circulation by the compression of the liver substance by the fibrosed capsule in contracting and the slight subcapsular cirrhosis. Pick (1896) ascribed the ascites to the liver; he believed that as a result of the pericarditis and adherent pericardium there was an interference with the return circulation which, long continued, gradually produced a congestive or cyanotic induration of the liver, and ascites followed in due course. The inflammation and thickening of the capsule of the liver are always marked, and it is reasonable to assign to this the greater share in the production of the ascites. The liver in these cases is usually adherent to the diaphragm, as are also the lungs. The movements of the diaphragm are reduced to a minimum and in addition the channels of communication through it are blocked. In polyserositis, the liver is at first enlarged, then gradually becomes smaller through compression by contraction of the thickened fibrous capsule.

Clinical Features and Diagnosis

The symptoms of polyserositis are variable and the condition may be latent for years or be preceded by inflammation of one or more of the serous membranes. While the clinical picture may be benign, its varieties depend upon the site of origin of the serositis and the manner of its development.

Polyserositis is essentially a disorder of the early adult or middle periods of life. Typically there is a chronic obliterative pericarditis without valvular disease of the heart; enlargement of the liver with perihepatitis; a constantly recurring ascites and little or no œdema of the legs and ankles. The condition begins in one serous membrane, more commonly in the pericardium than any other. The course is slow; and spread from one serous membrane to another is often found without signs that are distinctive. Often the patient is first seen when ascites has become well established. As the early stages affect serous membranes alone it is often difficult or impossible to trace the onset unless effusions form early or quickly. There are reasons sometimes for thinking that the disease may have existed for years before the patient seeks advice.

Thus the development of symptoms is prone to be insidious. There may be vague constitutional disturbance and smouldering pyrexia. There may be abdominal pain due to involvement of the peritoneum; or there may be recurrent effusions into serous cavities, requiring frequent tapping—much more often than is necessary in cirrhosis of the liver or simple pleurisy with effusion. Jaundice is usually absent although I have seen it appear early. There may be obstruction to the great veins of the trunk with œdema of the limbs. And yet in spite of such features reasonably good health may be retained for as long as five to twenty years.

Ascites is often the first indication of the disease, and although recurrent, in the course of time this may cease. Rumpf's (1895) case was tapped 301 times. Osler (1896) describes a child who was tapped 121 times. Sometimes the presenting symptoms are those of a failing heart, palpitation, dyspnœa, cough, cyanosis and generalised œdema; then ascites is a much more outstanding feature than in cardiac failure from other causes. Pain, which is fairly common, may be anginoid in character.

From what has been said already the difficulties with diagnosis are apparent. Polyserositis should be considered as a possibility in the presence of any of the following clinical pictures: mediastinal or abdominal venous obstruction; recurrent effusions into serous cavities; enlargement of the liver. The chief conditions from which it needs to be distinguished are mediastinal tumour, Hodgkin's disease, adherent pericardium, cirrhosis of the liver, certain forms of visceral syphilis, tuberculous peritonitis, chronic phases of rheumatic fever, and chronic splenitis of the splenic anæmia type.

Pick (1896) was the first to differentiate cases of polyserositis from cirrhosis of the liver. If there is a history of previous pericarditis and signs of an adherent pericardium are distinctive enough, then diagnosis from cirrhosis of the liver is not difficult. If there is no history of value, a careful physical examination may still enable a distinction to be made. In polyserositis, too, the associations of cirrhosis of the liver are absent. Polyserositis usually occurs

in young people. There may be no jaundice or signs of congestion of the portal system and the liver is usually enlarged. The ascites is much greater than in cirrhosis and requires frequent tappings. In polyserositis the patient may feel well and may live for years. In cirrhosis of the liver, as Hale-White (1908) pointed out, once ascites has set in the patient does not long survive to require further tappings. The presence of a recurring ascites in a person who is otherwise in good health and who is ambulant between tappings suggests at once the possibility of polyserositis. The ascitic fluid may be of an inflammatory type; and if there is, in addition, some pleural or pericardial involvement with thickening of these membranes or effusions into the cavities and a slow, protracted or intermittent course, the diagnosis of polyserositis may be made with some confidence.

Course, Prognosis and Treatment

Though polyserositis may begin as an acute process, more often it is insidious and obscure, with little clinical evidence. The course of the disease is extremely chronic, and punctuated by remissions of comparative good health. The prognosis for eventual recovery is poor, though about 70 per cent. of cases survive two years or more and about 50 per cent. four years or more.

The treatment is wholly symptomatic. In the acute stage absolute bed-rest is advisable, and pain, dyspnœa and cough require relief by sedatives and anodynes. Many remedies have been tried in hope of limiting the inflammatory process or restricting the accumulation of fluid. Cardio-tonics, salicylates, guaiacol carbonate, cod-liver oil, calcium lactate, potassium iodide, salines and various diuretics have all been tried, but without any results of value. A salt-free diet has also failed to have much influence, and has not been found to be worth the trouble. Tuberculin also has had little success.

More recently the newer mercurial diuretics, given either intra-muscularly or intra-peritoneally along with ammonium chloride by mouth, have been followed by an effective diuresis and an immediate general improvement in distressed patients. Noth (1937) reports a case treated for eight years with salyrgan, ammonium nitrate and abdominal paracentesis. This patient received about 450 doses of salyrgan and abdominal paracentesis was performed on more than forty occasions. Other than this, tapping the abdomen is our main resource in recurring ascites. To delay the re-accumulation of fluid, adrenalin in 1-1,000 solution may be injected intraperitoneally. The fluid is first withdrawn and 15-20 minims of adrenalin are injected daily.

Surgically little of a curative nature can be done for the relief of the condition. One operation suggested is the breaking up of peritoneal adhesions to establish a collateral circulation between the liver and the abdominal parietes, as advocated by Talma-Morison in cases of cirrhosis of the liver with ascites. Perhaps some of the reported Talma-Morison operations have been performed on patients with polyserositis in the belief that the condition was uncomplicated cirrhosis of the liver. In the event of the condition being tuberculous, the mere opening of the abdominal cavity would probably have been beneficial.

Operative procedures now used with success for chronic constrictive

pericarditis hardly come within the scope of the present paper. But one point must be made: A patient with active polyserositis and caseous changes in the pericardium is not suitable for an exploratory operation, let alone for pericardectomy; and if this operation is undertaken in the false belief that he has a true constrictive pericarditis with secondary mechanical effusions, the result is likely to be disastrous.

My thanks are due to Dr. Constant Ponder and to Dr. F. J. Pierce for their kind permission to publish this paper, and for giving me free access to the case-records at the sanatorium. I am very grateful to Dr. D. H. Mills for much help and constructive criticism.

REFERENCES

Boyd, W. (1931): "Pathology of Internal Diseases," London.
Cabot, R. C. (1898): *Boston Med. and Surg. J.*, 138, rye.
Chappelle de la, C. E., and Graef (1933): *Med. Clin. N.A.*, **17**, 147.
Concato, L. (1881): *Gior. internaz. d. sc. med., n.s.*, **3**, 1037.
Curschmann, H. (1884): *Deutsche Med. Wchnschr.*, **10**, 564.
Dawd, C. N. (1923): *Ann. Surg.*, **77**, 423.
Erskine, E. B. (1935): *Naval Med. Bull. Wash.*, **33**, 103.
Evans, G. H. (1918): *J. Amer. Med. Sci.*, **155**, 553.
Fagge, C. H. (1875): *Guy's Hosp. Rep.*, **20**, 55.
Guyer, O. K. G., and Smith, F. B. (1936): *Lancet*, **1**, 362.
Hale-White (1908): Allbut and Rolleston, "System of Med."
Herrick (1902): *Trans. Chicago Path. Soc.*, April.
Howard, T., and de Veer, J. A. (1936): *Amer. Rev. Tuberc.*, **33**, 755.
Kelly, A. O. J. (1903): *Amer. J. Med. Sci.*, **125**, 116.
Kraus, R., and Hofer (1912): *Deutsche med. Wchnschr.*, **38**, 1227.
Kraus and Willis (1926): *Amer. Rev. Tuberc.*, **14**, 316.
Laennec (1819): "Traité de l'Auscultation Med et des Maladies des Poumons et du Cœur," Paris.
Lenoble, E., and Pineau A. (1927): *Bull. et mém. Soc. Méd. d'Hôp. de Paris*, **51**, 130.
Mallory (1929): *New Eng. Med. J.*, **201**, 588.
Mannering, W. H., and Bronfenbrenner, J. (1913): *J. Exp. Med.*, **18**, 601.
Matthew, E. (1912): *Edin. Med. J.*, **9**, 310.
Mayo, W. J. (1922): *Ann. Surg.*, **76**, 432.
Mosler, E. (1910): *Med. Press*, **90**, 433.
Much, H., and Leschke, E. (1911): *Beitr. z. Klin. d. Tuberk.*, **20**, 405.
Nicholls, A. G. (1902): *Studies from Royal Vict. Hosp. Montreal*, 1, no. 3.
Nicholls, A. G. (1903): *J. Amer. Med. Assoc.*, **40**, 696.
Noth, P. H. (1937): *Proc. Staff Meet. Mayo Clinic*, **12**, 513.
Osler, W. (1896): "Practice of Med." N.Y., p. 625.
Osler, W., and Macrae (1926): Art. by Kelly, rev. by Lyon, **3**, 783.
Patterson, R. C. (1917): *Amer. Rev. Tuberc.*, **1**, 353.
Paul (1930): *Bull. Mayo Clin. Lab.*, **9**, 16.
Picchini, L. (1891): *Il Morgagni*, **30**, **3**, 608.
Pick, F. (1896): *Ztschr. Klin. Med.*, **29**, 385.
Pick, F. (1899): *Verlandl. d. cong. f. innere Med. Wiesb.*, **17**, 588.
Pick, F. (1900): *Wien Klin. Wschnschr.*, **13**, 324.
Ramsey, T. L. (1937): *J. Lab. Clin. Med.*, **23**, 39.
Reed, A. (1935): *Southern Med. J.*, **28**, 729.
Reid, W. D. (1920): *Bost. Med. Surg. J.*, **183**, 386.
de Renzie (1898): cited by Hager, *J. Gesellschaft zu Magdeburg*, 39.
Rist, E., and Holland, J. (1914): *Ann. de Med.*, **2**, 13.
Rohde, C. (1927): *Deutsche Ztschr. f. Chir.*, **203**, 18.
Rokitansky, C. (1842): *Handbuch der speciellen Pathologischen Anatomie*, **2**, 317.
Rolleston and McNee, J. W. (1912): "Dis. Liver and Bile Ducts," Edin.
Rosenbach, O. (1907): Cornet, "Die Tuberkulose," **2**.
Rumpf, H. (1895): *Deutsch. Arch. f. Klin. Med.*, **55**, 272.
Smith, W. H. (1913): *J. Amer. Med. Assoc.*, **61**, 739.

STOLL, H. F. (1929): *J. Amer. Med. Assoc.*, **92**, 791.
STONE, W. J. (1926): *Amer. Heart J.*, **1**, 434.
TISSIER (1885): *Le Progrès Médical*, **8**, 234.
VOLHARD and SCHMEIDEN (1923): *Klin. Woch.*, **2**, 5.
WILKS, S., and MOXON, W. (1875): Lect. on Path. Anat., London.
YING, Y. Y. (1936): *Inter. J. Leprosy*, **4**, 487.

AN UNUSUAL COMPLICATION OF GOLD THERAPY

BY B. G. RIGDEN.
From Tor-na-Dee Sanatorium, Aberdeenshire.

THOUGH treatment by means of gold salts is often hampered by the development of muco-cutaneous changes or gastro-intestinal disturbances, it is less common to meet neurological complications. A survey of the literature suggests, however, that the latter are by no means rare, and that they take the form of polyneuritis or radiculitis, often associated with insomnia. Mental disturbances seem to be encountered comparatively seldom. Widespread fibrillary twitchings and insomnia were the chief features of cases described by Chavany and Chaingot (1934) and by Gernez and Nayrac (1934). Insomnia was also a feature of the cases reported by Devic and Bouquin (1935), by Bourrat (1935), and by Bernard and Morin (1936). The case of the last-named authors again showed fibrillary twitchings. Devic, Mansuy and Ronsard (1936) describe a sensory polyneuritis, associated with anxiety, irritability and insomnia, and consider this syndrome to be the commonest neurological complication. Spangenberg, Munist and Ardaiz (1937), Hoelen (1940) and Pearson (1940) report further cases where insomnia was a prominent feature.

In a most interesting paper Pereyra Käfer (1940) gives a full review of the subject. He states that complications may arise at any time during the course of treatment, when he attributes them to gold intolerance, but that they usually occur after the termination of injections, when they are most probably the result of true gold poisoning, the body having accumulated an abnormal amount of the salt. Further observations are that nervous complications are usually associated with muco-cutaneous, renal or hepatic changes, and that the prognosis in cases with mental disturbance, even where the upset is severe, is good. This last conclusion is borne out by an examination of the reported cases, of whom very few have died. Sunderlin (1941) reviews 730 cases of chronic arthritis treated with 1,095 courses of gold injections, and states that out of 694 complications 107 affected the nervous system. In ten there were mental symptoms amounting to a psychosis. Two cases were fatal; the others recovered completely.

Case Report

Miss M., aged 39, was admitted to the sanatorium in 1937 when she was found to be suffering from an acute tuberculous lesion in the left lung. There was also laryngeal involvement. Following induction of a left artificial pneumothorax, the disease spread to the right lung, so that a second pneumo-

thorax had to be established on that side. While the larynx healed swiftly and permanently the pulmonary disease did not respond so well. Effusion developed at various times on both sides and it was not possible to keep the right pneumothorax going for very long. The left A.P. proved more satisfactory and is still being maintained. After a long spell of treatment the patient eventually became quite well stabilised and she was able to leave the sanatorium in August 1941. She did very well for the next four months, but was unfortunate enough to relapse in December. At that time she was re-admitted to the sanatorium with a high temperature and other signs of toxæmia. Although the temperature soon settled after a spell of bed-rest the condition as a whole remained rather unsatisfactory, little progress being made. The patient never seemed quite able to get on top of her disease and she seldom felt at all fit.

In the late summer of 1942 there was a new extension of disease in the right lung, for which it was decided to try a course of intramuscular gold injections. These were started in November and were continued in cautious dosage until, by January 19, 1943, a total of 1·48 grammes of Myocrisin had been given. At this stage the injections were discontinued on account of stomatitis, the first complication to develop and one that soon subsided. At about the same time the patient complained of an irritable dermatitis: this persisted for several weeks and was very resistant to every measure devised for its relief.

On February 4 signs were first noticed of emotional disturbance, the patient being unwilling to receive visitors, an unusual trait for her to exhibit. Mental instability became more evident during the next few days, the lady becoming unwontedly garrulous and experiencing alternate fits of excitement and depression. On the 6th she first complained of nausea and of headache, and she refused all forms of food. By the following day the headache had become much more severe and the patient was very drowsy. On entering the room one was faced by a striking picture: the patient lay curled up on her left side with her face turned away from the dim light admitted by the shaded window. Her head was bent forwards and was partially buried in the pillows, while her legs were drawn up to her abdomen. When, after some little difficulty, she was roused, there was mild confusion and disorientation. As the patient was obviously very ill examination was perforce restricted to a minimum, but definite neck rigidity and photophobia were observed, while the knee-jerks were brisk and the plantar responses were flexor. The clinical appearances were typical of tuberculous meningitis and a specimen of lumbar fluid was withdrawn in the confident expectation that its examination would confirm the bedside diagnosis. In the event, however, the fluid showed no abnormal characteristics whatever. A blood-urea estimation and a white cell count both gave normal results.

By February 12 the condition had improved sufficiently to allow a rather fuller examination to be made. On that date there was no abnormality to be found on examining the cranial nerves. The pupils were equal, central and circular, reacting normally to light but accommodating only sluggishly. The knee-jerks were less brisk than formerly and the plantar responses were still flexor. The mental state remained changeable, with excitement predominating over depression, while mild confusion, overshadowed by drowsiness, persisted during the periods when the patient was not asleep. By this time she was able to take fluids well and she was accordingly given frequent glucose drinks, with large doses of vitamins B and C.

Thereafter improvement was much more rapid, so that by the end of

February the patient was once more her old self. The pulmonary condition was entirely unaffected by the upset.

Comment

The salient feature of this case was that the clinical condition so strikingly resembled tuberculous meningitis. The other remarkable aspect was that the condition was characterised by drowsiness rather than by insomnia, the latter being a common finding in cases of gold poisoning or intolerance.

As the complication developed fairly early during the course of treatment, before a large amount of gold had been given, it would seem that it was due to intolerance rather than to true gold poisoning.

Summary

A short review of the literature shows that neurological complications of gold therapy are fairly frequently encountered. Mental disturbances, on the other hand, are comparatively rare.

A case is described where a patient receiving gold salts for the treatment of pulmonary tuberculosis developed a condition clinically resembling tuberculous meningitis. The cerebro-spinal fluid showed no abnormality, however, and the patient made a rapid and complete recovery.

I wish to express my thanks to Dr. R. Y. Keers for his helpful advice in preparing this paper.

REFERENCES

BERNARD and MORIN (1936): *Bull. Soc. méd. Hôp. Paris*, **52**, 469.
BOURRAT (1935): *Lyons méd.*, **156**, 517.
CHAVANY and CHAINGOT (1934): *Presse méd.*, **42**, 478.
DEVIC AND BOUQUIN (1935): *Lyons méd.*, **156**, 406.
DEVIC, MANSUY and RONSARD (1936): *Lyons méd.*, **157** 473.
GERNEZ and NAYRAC (1934): *Rev. Neurol.*, **41**, 839.
HOELEN (1940): *Ned. Tijdschr. Genesk.*, **84**, 1514.
PEARSON, S. VERE (1940): *Tubercle*, **21**, 313.
PEREYRA KÄPER (1940): *Sem. med. B. Aires*, **2**, 358 and 420.
SPANGENBERG, MUNIST and ARDAIZ (1937): *Prensa med. Argent.*, **24**, 1386.
SUNDERLIN (1941): *Acta med. Scand.*, suppt. 117, 69 and 180.

MEETINGS OF SOCIETIES

YORKSHIRE TUBERCULOSIS SOCIETY

SESSION 1942-43

THIS Society, which, like many others at the beginning of the war, showed a temporary loss of function, has regained full activity again, and the session just completed has been one of the best in the history of the Society. The meetings, held in the Leeds Medical School, have all been well attended.

Miniature radiography was discussed very ably by Major Peter Kerley, who showed the disadvantages as well as the advantages of the method, and emphasised the need for faultless technique from a team whose training must be first-class. Equipment must also be 100 per cent. reliable, but the success

or failure of mass radiography depends, more than ever, on *interpretation*. In Major Kerley's survey, 3 per cent. of the miniatures were suspect, of which, after full X-ray examination, half were discarded, so that 1·5 per cent. of cases were sent for clinical examination. Disappointments of miniature radiography are: (1) that early pulmonary tuberculosis is not revealed in the film, and (2) the X-ray diagnosis of *healed* pulmonary tuberculosis is unreliable. The co-operation of radiologist and tuberculosis officer is most desirable in the ultimate success of any scheme of mass miniature radiography, and the snags will more readily be avoided if this co-operation is well-founded and long maintained.

"Bilateral Pneumothorax Treatment" was the subject of a stimulating paper by Dr. Tattersall, who wished that sanatoria would be more ready to try this out, as he was convinced that many more cases of pulmonary tuberculosis would benefit by it than were at present receiving collapse therapy. During the discussion some interesting points about the position of the patient during refills were raised, and Lieut. George Day provided some films to show the influence of posture on the relative position of lungs, mediastinum and diaphragm.

" The Differential Diagnosis of Pulmonary Tuberculosis " was discussed by Dr. Moll, who, in a very sound analysis of signs and symptoms, covered the field of diagnosis most thoroughly.

The stimulus towards collaboration between radiologists and clinicians was developed by the formation during the session of a " Radiologists' Brains Trust," composed of Major P. Kerley, Dr. A. S. Johnstone and Dr. A. Pollitt. At the first meeting of this kind, members of the Society provided the films, to which the questions were attached, with or without clinical details, and the radiologists provided the answers. At the return meeting the radiologists provided the films, *without* clinical data, for the clinicians to diagnose, and a very merry time was had by all ! Certain deductions might be drawn from these experiments, which, being as yet " not proven," will, we hope, be defined by further trials of strength between the two protagonists. There is no doubt at all about the benefits accruing, because all members can participate, and the idea may appeal to other Societies, not only in the tuberculosis world. The relative importance of the clinical and X-ray diagnosis will become clearer.

REVIEWS OF BOOKS

Brompton Hospital Reports, vol. xi., 1942.

The articles which comprise the latest volume of these Reports are well up to the standard which we have come to expect of them. The list of contributors contains many names well known in the thoracic world, and the choice of subjects is a varied one. All branches of intrathoracic work are represented, and there is a welcome article from the lady almoner's department. The co-operation of this department is perhaps more valuable in the war against pulmonary tuberculosis than in any other field of social medicine.

The book opens with a fitting tribute to the late Dr. Percy Kidd, who was first appointed to the staff of the Brompton Hospital in 1881, and ends with an outstanding article on the care of the dying. The correct and intelligent use

of drugs is an item of medical teaching which is for the most part entirely neglected today, with the result that there is growing up a generation of doctors having little skill in the use of that essential weapon of their armament.

" The Anatomy of the Bronchial Tree " is a most original and instructive paper, based on a personal study of over two hundred bronchograms, assisted by bronchoscopic observations, post-mortem specimens and bronchial casts. The many important branches of the bronchial tree are described and a new terminology is suggested. The result is probably the most accurate description of the bronchial tree yet published in England.

War surgery is represented by a paper on chest injuries, in which the essentials of treatment are laid down. The factors which demand such treatment—*i.e.*, interference with the cardio-respiratory reserve in the early case, and sepsis in the late case—are admirably stressed.

The same author has also written a controversial paper on the mechanics of lung cavities—why some collapse and other apparently similar ones actually expand. The supposition is that inherent retractibility of lung tissue, counteraction of chest wall pull by compensatory emphysema, and bronchial stenosis are the three most important factors concerned.

Another highly controversial subject is discussed in an article on the pathogenesis of pulmonary tuberculosis of the adult type. The author sets down simply and briefly what are generally considered to be proven facts, and advances an attractive hypothesis of his own—that exogenous superinfection causes endogenous exacerbation.

The diagnosis, treatment and prognosis of the paravertebral abscess which ruptures into pleura or lung is carefully considered in an article well illustrated by case histories and X-rays. Six such catastrophes are discussed and emphasis is laid on the importance of pleural pain and a non-productive cough as prodromal symptoms of this disaster. Frequent aspiration, and even free surgical drainage of the abscess, is suggested when rupture is judged to be imminent.

Mass Miniature Radiography. By R. R. TRAIL, H. J. TRENCHARD, and J. A. KENNEDY. Published by J. and A. Churchill, Ltd., of London. Price 8s. 6d.

This little book has been written as a guide to the setting up of a mass miniature radiography centre, and its management. It is based on the experience of the authors in dealing with 150,000 cases, and the follow-up of the abnormal ones.

Administration, apparatus, and buildings are fully described in the earlier parts of the book, and are followed by some suggestions for staffing a department. Detailed recommendations are made for X-ray and dark-room layouts, with some remarks on the processing of 35 mm. fluographic film. The authors' methods of running a department and viewing films are described, and an interesting section on interpretation of miniature films is included, together with the reproduction of a number of illustrative miniature films and their case-histories.

The authors succeed in demonstrating the paramount importance of meticulous administration and detailed clerking, and their book will prove of value to medical officers and radiographers who are about to begin work in a mass miniature radiograph unit.

Rehabilitation of the Tuberculous. By H. A. Pattison, M.D., F.A.C.P. The Livingstone Press.

The author of this book from America has made a study of rehabilitation as it applies to the tuberculous patient. He has realised the enormously important part that after-care must play in the lives of those suffering from pulmonary tuberculosis. But it cannot be said that the book is worthy of one who must have a vast knowledge of tuberculosis as it affects the mind and the body.

The book contains much of value, but the manner of its telling leaves a great deal to be desired. The English is bad, and punctuation is scanty. For the physician the book is not scientific enough or detailed enough: for the layman there is much in it which must be difficult to understand, particularly the quotations of various authors in the chapter on the human constitution. In spite of these inadequacies the book serves a good purpose in drawing attention to one side of tuberculosis therapy which is not sufficiently stressed. A vast proportion of the world's tuberculous patients are still treated in sanatoria, and the more fortunate ones, whose disease has been arrested, are flung back into an unsympathetic world to earn a living as best they can. Most of them are dead within five years because no provision, either monetary or occupational, is made for them during the all-important period immediately following their discharge.

The chapters on the mental aspects of tuberculosis, and on marriage of the tuberculous, are good. The ideal physical criteria are balanced against the hopes and fears of human nature, and no one would deny the wisdom of the resulting conclusions.

Rehabilitation colonies in the different countries are described in the second part of the book. The work of these settlements is for the most part successful, in that persons with comparatively quiescent disease are trained and given a trade which enables them to earn a living, whilst working under medical supervision. But the numbers catered for are an infinitesimal fraction of the tens of thousands of patients throughout the world, for whom an expensive sanatorium treatment means no more than a prolongation of life for a further three or five years.

The subject is an enormous one and a most important one. It is to be hoped that social medicine of tomorrow will give it the attention it so richly deserves.

Summary Report of the Ministry of Health for the year ended 31st March, 1943. H.M. Stationery Office. Price 1s. Cmd. 6468.

This Report sums up the present state of the public health in England and Wales in these words: "The best judgment that can be reached on the position at the end of 1942 is that, according to all the tests that were applied before the war, the general health of the nation has been remarkably well maintained. While there is some evidence of an increase in minor and short-term illness, this did not lead to an increase in illness of a more serious type or longer duration; and minor illness appears to have been no more prevalent than might have been expected at this stage of a hard war."

1942 was a year of record-breaking in vital statistics. Not only the maternal and infant mortality rates, but also the proportion of stillbirths, and the standardised death rates amongst civilians, both male and female, were the lowest ever recorded in England and Wales. The incidence of infectious diseases was remarkably low—probably the best on record.

The Report points out that although statistics provide the basis for strict comparison with the years of peace, and afford reliable evidence of the general well-being of the population, they cannot reflect the health situation in detail, or measure " positive " health; and minor ailments and the " below par " or " tired " feeling do not come within their scope. There is, in fact, no simple way of measuring the health (in a positive sense) of forty million people, though existing methods are constantly being extended.

" For example, a number of nutritional surveys were made in 1942. They showed that there has been no general deterioration in the nutritional state of the population. On the other hand, sample enquiries among doctors, considered in conjunction with the rising claims to sickness benefit under the National Health Insurance Scheme, suggest that there was a considerable increase in short-term sickness during the year. . . . An increase in minor illness might well be expected after more than three years of war, with all its anxieties; long hours of employment, often on heavy and unusual work; shopping, housing and travelling difficulties; Civil Defence or Home Guard duties; lack of holidays; and the black-out. There is no indication of any increase in long-term illness "—a reassuring statement for the harassed and overworked clinician to digest.

For many groups of diseases, new low records in the number of deaths were established. These include pneumonia, influenza, diphtheria, scarlet fever, rheumatic fever, gastric ulcer and other diseases of the stomach. Deaths from influenza (3,401) were fewer by 3,500 than in 1941. " There can be little doubt," says the Report, " that sulphonamide therapy has been responsible in large measure for the decline since 1938 in deaths from a number of these causes, in addition to saving the lives of many persons attacked by cerebro-spinal fever during 1942." The principal diseases showing an increase in deaths compared with 1941 include cancer (though the standardised death rate showed no rise), Hodgkin's disease, coronary disease, angina pectoris, enteritis and diarrhœa.

With regard to the second of two main dangers, tuberculosis, " the position in the third year of war was better than could reasonably have been expected two years ago." There was a distinct check in the wartime increase in the death rate. Total number of deaths from all forms of tuberculosis was 25,547—about 2,500 less than in 1940, 3,000 less than in 1941, and much the same as in the last pre-war year (1938). (The 1938 figure was the lowest on record and well under half the corresponding figure for 1918.) " While these figures give ground for satisfaction," says the Report, " vigilance must be maintained. There was a rise of about 30 per cent. in the number of new cases reported last year, and under the trying conditions of total war it is not unlikely that the death rate will tend to rise again. . . . Two important forward steps have been taken. First, the newest method of chest examination by miniature X-ray photography was introduced, though as yet on a limited scale. Secondly, a scheme was initiated for the payment of special allowances to people who leave remunerative employment to undertake treatment for pulmonary tuberculosis, to relieve them from anxiety about those who look to them for support and upkeep of a home."

Other sections of the Report deal with mothers and children, housing and the Emergency Medical Service. We are left with the feeling, after reading this Report, that the Browning belief in supernatural optimism is one in which our present health authorities are certainly well schooled.